Case Report

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Brain abscess in a 12-year-old child with congenital heart disease: lessons learnt

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ABSTRACT

Brain abscess is a rare and often life-threatening sequelae, observed in 5-20% of the patients with cyanotic congenital heart diseases (CHD). Cyanotic heart diseases involve a right-to-left shunt by-passing the pulmonary vascular bed, culminating in tissue hypoxia. We present a case of an uncorrected atrioventricular canal defect (AVCD) with a double outlet right ventricle (DORV) complicated by a brain abscess in a 12-year-old girl. This patient was a known case of CHD diagnosed at 2 years of age and presented to the neurology out-patient department with a holocranial headache, vomiting, and low-grade fever for 5 days. General examination was suggestive of cyanosis and low-set ears. Systemic examination revealed a pansystolic murmur with bilateral extensor plantar reflexes. MRI brain with contrast revealed an abscess in the right temporoparietal lobe with perilesional edema. The patient was managed conservatively. Nine days post-discharge, the patient presented with generalized tonic-clonic seizures and was readmitted and managed accordingly. Subsequently, she was referred to the cardiothoracic surgery department for correction of the CHD. However, the family gave negative consent for the same. The relatives of the case described here were counseled to get a surgical correction done for the cardiac defect at the time of diagnosis but with no success. The aim of publishing this case is to emphasize the need for timely diagnosis and correction of such anomalies to curb serious complications causing profound morbidity and mortality.

Keywords: Brain, Abscess, CHD, Cyanotic, Hypoxia

INTRODUCTION

A brain abscess is a localized collection of pus enclosed in the brain tissue, usually caused by a bacterial or fungal infection.1 It can spread via direct local spread from the site of infection such as paranasal sinusitis, otitis media, mastoiditis, or dental infection. It can also develop post head injury, through neurosurgical intervention, or via a hematogenous route. Comorbidities including diabetes mellitus, cirrhosis of the liver, history of stroke, HIV infection, and autoimmune disorders may also predispose to the occurrence of brain abscesses.²

The patient usually presents with headache, fever, convulsions, altered mental status, nausea, vomiting, and focal neurological deficits. Commonly

incriminated organisms include Streptococci, Enterobacteriaceae, anaerobes, Staphylococcus, and fungi (Candida, Cryptococcus and Aspergillus).3

Brain abscess can lead to several complications such as new onset seizure activity, recurring abscess, meningitis, and focal neurological deficits (hemiparesis, aphasia, or visual field defects). 4 Diagnosis of brain abscess includes neuroimaging (MRI superior to CT scan). Additional supportive investigations include gram stain and culture of abscess material, complete blood counts, ESR, Creactive protein, and blood cultures.

Treatment involves high-dose parenteral antibiotics with or without neurosurgical drainage.

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A quarter of brain abscesses occur via the hematogenous route.⁵ Cyanotic CHD s lead to hypoxia that is caused as a result of right to left shunts bypassing the pulmonary vasculature, which results in hematogenous bacterial seeding and disruption of the blood-brain barrier permeability. It is an unusual but potentially lifethreatening complication, which can occur in 5-20% of the population with CHD.6 It is majorly due to chronic hypoxia resulting in lowered immunity polycythemia. The resultant hyper viscosity causes localized cerebral ischemia, providing a favorable environment for microbial proliferation. Corrective surgery for CHD early on in childhood can help decrease the risk of occurrence of a brain abscess.⁷ Although rare. brain abscess is also seen in specific CHDs like DORV and AVCD. Here we present a case of an uncorrected AVCD with a DORV complicated by a brain abscess in a 12-year-old girl.

CASE REPORT

A 12-year-old girl was admitted to the hospital with a diagnosis of a brain abscess. She presented with a history of headache, vomiting, and fever persisting for 5 days. The headache was described as holo-cranial, continuous, and moderate in intensity. The patient had experienced 3-4 episodes of non-bloody and non-bilious vomiting along with low-grade fever without chills and rigors, lasting for 2 days. Notably, she had been diagnosed with congenital heart disease, specifically AVCD with DORV, at the age of 2 years but had not undergone any surgical intervention despite medical recommendation. Additionally, she had a history of cyanotic spells.

On examination, cyanosis was evident, and the patient exhibited a low set of ears. Her oxygen saturation (SpO₂) remained around 84% on room air. Neurological examination revealed a Glasgow coma scale (GCS) score of E4 M6 V5 with bilaterally extensor plantar reflexes. However, she was conscious, responsive to voice commands, and demonstrated normal tone and power in all four limbs. Pupillary examination showed bilaterally equal reactivity to light. A cardiac examination revealed a pan systolic murmur, while other heart sounds were normal. The remainder of the systemic physical examination was unremarkable.

Laboratory investigations (Table 1) indicated polycythemia with a hemoglobin level of 21.8 gm/dl, hematocrit of 67%, and elevated inflammatory markers. Blood and urine cultures were negative for microbial growth.

MRI brain contrast (Figure 1) revealed a well-defined approximately 30×27×21 mm (AP×TR×CC) sized peripherally rim enhancing altered signal intensity lesion with perilesional edema in the right parietal lobe, indicative of intraparenchymal abscess with intraventricular extension and features of meningitis and ventriculitis.

Table 1: Hematological investigations at the time of initial admission.

Investigations	Results	Reference range
Hb (g/dl)	22	12-18
TLC (/µl)	7900	4000-10000
APC (/μl)	230000	150-410
Urea (mmol/l)	43.8	15-40
Serum creatinine (mg/dl)	0.28	0.6-1.2
Na+ (mEq/l)	135	135-145
K+ (mEq/l)	4.74	3.5-5.5
SGPT (U/I)	30	10-49
Total/direct bilirubin	0.82	0.3-1.2/
(mg/dl)	/0.3	0-0.2
Total proteins/serum	5.76/	5.7-8.2/
albumin (gm/dl)	3.19	3.2-4.8
CRP (mg/l)	86	<5
PCT (ng/ml)	11.8	< 0.05

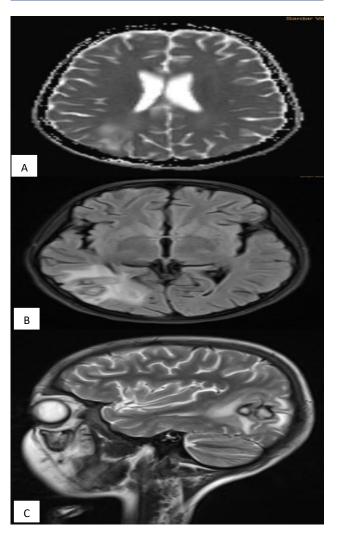


Figure 1 (A-C): MRI brain with contrast of well-defined approximately 30×27×21 mm (AP×TR×CC) sized peripherally rim enhancing altered signal intensity lesion with perilesional edema in right parietal lobe.

A: Transverse view; B: Coronal view and C: Sagittal view.

X-ray chest was unremarkable. (Figure 2) Echocardiography findings were consistent with situs solitus, along with the presence of common complete AVCD, DORV, severe infundibular and valvular pulmonary stenosis, mild atrioventricular (AV) valve regurgitation, large ostium primum atrial septal defect (ASD), ventricular septal defect (VSD), and both great vessels arising from the right ventricle with normal AV concordance.



Figure 2: X-ray chest postero-anterior view.

The patient was initially managed conservatively with intravenous antibiotics including ceftriaxone, vancomycin, and clindamycin. Due to thrombocytopenia, ceftriaxone was replaced with cefepime, and the regimen was continued for 6 weeks. Follow-up imaging showed a mild reduction in abscess size and edema, and the patient was discharged with oral antibiotics for an additional 7 days.

Nine days post-discharge, the patient presented with generalized tonic-clonic seizures and was readmitted and managed accordingly. Subsequently, she was referred to the Cardiothoracic surgery department for correction of the CHD. The patient has been on medications, including anti-epileptics, for the past year. Despite her longstanding history of CHD, she has not undergone any surgical intervention to date.

DISCUSSION

This case of a 12-year-old girl with a brain abscess in the setting of CHD specifically AVCD with DORV, presents a complex clinical scenario with several noteworthy aspects.

Brain abscess is a focal, suppurative intracerebral infection that begins as a localized area of cerebritis and

develops into a collection of pus surrounded by a well-vascularized capsule.

Etiologies include hematogenous spread from septic foci including infective endocarditis, pyogenic lung infections, urinary sepsis, and cyanotic CHD or direct spread from sources like sinusitis, otitis, or dental infections. Brain abscesses developing as a sequelae of cranial sites of infection (chronic sinusitis, otitis media, and mastoiditis) are the most common followed by those due to heart diseases, which constitute 5-20% of the total case burden.

The most common origin of brain abscesses in children is cardiogenic. Delayed diagnosis and treatment of cyanotic CHD lead to causation, resurgence, and relapse of cardiogenic brain abscess.⁶ Often these abscesses are multiple and have a preference for the middle cerebral artery territory (posterior frontal, temporal, and parietal lobes) followed by cerebellar and occipital lobes.⁹

Our case report describes the manifestation of a hematogenous brain abscess in a known case of CHD which, despite adequate medical management, complicated into new onset seizure activity. Often such abscesses are not amenable to medical therapy and may require neurosurgical intervention in the form of excision via craniotomy or craniectomy which are associated with their risks.

This stresses the importance of prompt diagnostic and surgical intervention in such cases right in childhood to decrease the morbidity and mortality burden and medical expenditure. Thus, the role of counseling family members of such patients and aiding them in early decision-making is the key to preventing this life-threatening disease and the complications that follow.

CONCLUSION

Hematogenous brain abscess is a rare and often lifethreatening sequelae of hypoxia associated with CHD. Often, further complications like recurrence, seizures, focal neurological deficits, and cognitive decline ensue following the occurrence of such abscesses. Early diagnosis and appropriate surgical correction of the underlying defect are of paramount importance in decreasing the morbidity and mortality burden as well as improving the quality of life in such patients.

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